Hyperacute T-waves Can Be a Useful Sign of Occlusion Myocardial Infarction if Appropriately Defined

Stephen W. Smith, MD*; Harvey Pendell Meyers, MD

*Corresponding author. E-mail: smith253@umn.edu.

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Koechlin et al, in this issue of *Annals*, using the APACE database, attempt to study the use of "hyperacute T-waves" in diagnosing acute myocardial infarction. Unfortunately, critical limitations in the definition of hyperacute T-waves and an outcome definition of any acute myocardial infarction make the results inapplicable to the concept of hyperacute T-waves seen in acute coronary occlusion myocardial infarction.

The authors start by emphasizing the need to act rapidly in ST elevation myocardial infarction (STEMI) to reperfuse acute coronary occlusion (occlusion myocardial infarction). Using the APACE database, they study 445 type I acute myocardial infarctions (including 82 STEMIs and 363 NSTEMI) and compare them to 2,012 patients without acute myocardial infarction. This author group previously reported that there were 135 occlusion myocardial infarctions among these 445 type I acute myocardial infarctions.[1,2] They have already shown in this cohort that STEMI criteria are not sensitive even for occlusion myocardial infarction, much less any acute myocardial infarction, as has been shown in many other studies.[3-6] It would indeed be appropriate to study whether hyperacute T-waves may identify occlusion myocardial infarction that ST elevation does not identify. This idea is especially timely because the American College of Cardiology and the American Heart Association recently formally endorsed hyperacute T-waves as a "STEMI equivalent" with the explicit goal of identifying acute coronary occlusion.[7]

After stating that patients with STEMI are the ones who need emergency ECG diagnosis, Koechlin et al hypothesize a very different idea: that hyperacute T-waves may be a marker of any acute myocardial infarction (STEMI + NSTEMI, or occlusion myocardial infarction + nonocclusion myocardial infarction) rather than only a marker of STEMI or occlusion myocardial infarction. This is a misunderstanding of the role of ECG in acute myocardial infarction. The ECG is not sensitive for Non-occlusion myocardial infarction, but it need not be sensitive because such patients do not need immediate reperfusion; they can wait for troponin to make the diagnosis of acute myocardial infarction. Similarly, we know that ST elevation identifies only a small fraction of all acute myocardial infarctions; this author group has already reported that the sensitivity of STEMI criteria for any acute myocardial infarction in this same APACE cohort was only 8%[1]. In spite of this knowledge, they hypothesize that T-wave amplitude alone may correlate with the diagnosis of any acute myocardial infarction. Therefore, the results are not surprising: T-wave amplitude in isolation poorly predicts the diagnosis of acute myocardial infarction (or even of occlusion myocardial infarction).

There is no research-based definition of hyperacute T-waves. Koechlin et al define them as high T-wave amplitude, disregarding other features of the T-wave or especially of T-wave size in proportion to the QRS size. We have recommended using proportional T-wave "bulk," defined by the increased area under the curve (AUC) of the T-wave relative to the QRS amplitude.[8,9] Subjectively, this corresponds to T-waves that are abnormally large by area (both height and width), bulky, and abnormally symmetric, as if being physically inflated, but all relative to QRS size. Objectively, increased AUC of the T-wave relative to the QRS has already been shown by our group, for left anterior descending artery occlusion, by the following 2 quantifiable elements: 1) longer corrected QT interval,[10,11] and 2) greater T-wave to QRS amplitude ratio.[10] An additional feature of hyperacute T-waves is increased and abnormal T-wave symmetry, which has not yet been well quantified (contrary to conventional wisdom, we have found that hyperacute T-waves are generally more symmetric than

normal T-waves). Each of these features corresponds with the overall T-wave "inflation" and increased AUC in T-waves of occlusion myocardial infarction. Our group compared subtle left anterior descending artery occlusion myocardial infarction to precordial benign early repolarization (normal variant ST elevation in lead V2-V4); we found that occlusion myocardial infarction had significantly longer QT interval than normal variant ST elevation. Moreover, this finding corresponds to balloon occlusion data showing that increased QT interval is the first easily measurable ECG indication of acute coronary occlusion.[10] Our group further showed that the T-wave amplitudes were identical between the 2 groups, but the R-waves in occlusion myocardial infarction were far smaller, and thus, the T-wave to R-wave amplitude ratio in V2-V4 was far higher in the left anterior descending artery occlusion myocardial infarction than the benign variant ST elevation.

Previously, Collins et al[12] have found that a ratio of T-wave amplitude to QRS amplitude of more than 75% was a strong predictor of myocardial infarction (though not distinguishing occlusion myocardial infarction from Non-occlusion myocardial infarction), establishing that proportions are more important than absolute amplitudes. Hochrein et al[13] studied patients undergoing thrombolytic therapy (therefore, these were patients thought to have occlusion myocardial infarction) and found that higher T-wave amplitude was a sign of better prognosis, as these patients were earlier in their presentation, and thus, the hyperacute T-waves were a sign that the ischemic myocardium was still viable.

Similar to the study of ST elevation, the study of hyperacute T-waves should be in correlation with the ischemic myocardial territory (anterior, inferior, lateral, and posterior). Koechlin et al simply reported the T-wave amplitude in all leads instead of the areas of the ECG corresponding to the myocardial territory at risk, which would require angiographic or echocardiographic correlation. If Koechlin et al, instead of studying T-wave amplitude, had studied ST elevation in isolation from the

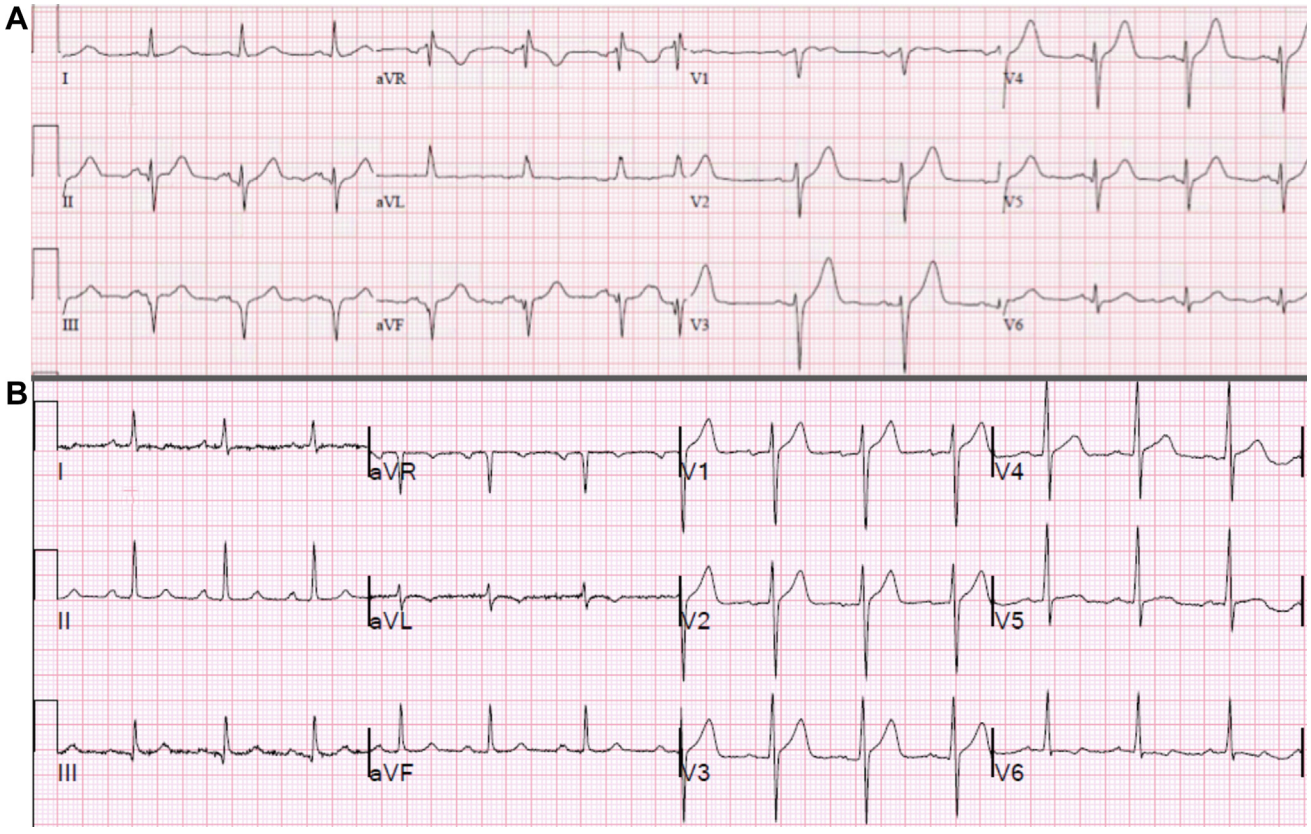

Figure 1. Figure 1 shows two cases from our database, chosen simply to illustrate the effect of different definitions of hyperacute T-waves. The top panel shows the ECG of a Patient A with proven acute LAD occlusion, while the bottom panel shows Patient B who ruled out for AMI with three serial negative troponins and a final diagnosis of noncardiac chest pain. Notice that patients A and B have similar T-wave amplitude in precordial leads, and patient B has more ST elevation than patient A. Although the amplitude and AUC of the T-waves in V2-V4 is nearly equal, the Occlusion MI in patient A is distinguished by an abnormally increased ratio of T wave area to the R wave and QRS size, and also because the T-waves are abnormally symmetric.

ischemic territory, it too may not have shown an association with acute myocardial infarction. Accordingly, when Hochrein et al[13] studied isolated T-wave amplitude (not in proportion to the QRS) in the same leads as ST elevation in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries trial, they found that "tall" T-waves were indeed an independent marker of benefit from thrombolytics, such that patients with STEMI and tall T-waves who received thrombolytics had a lower 30-day mortality than patients with STEMI without tall T-waves, and were less likely to develop heart failure and cardiogenic shock.

When hyperacute T-waves are considered with the appropriate definition and outcome measures, they can be a very important feature of occlusion myocardial infarction. We studied occlusion myocardial infarction ECG findings in 808 high-risk patients with a potential acute coronary syndrome (comprising 3,421 ECGs), with a 49% rate of acute myocardial infarction and a 33% rate of occlusion myocardial infarction.[3] Among other findings, expert ECG interpreters documented the presence or absence of hyperacute T-waves in all leads of all ECGs, blinded to the clinical outcome. Expert ECG interpretation was found to be superior to STEMI criteria for the identification of occlusion myocardial infarction, and we noted hyperacute T-waves in 72 of 146 patients (49%) who were correctly identified as having an occlusion myocardial infarction several hours before the occlusion myocardial infarction was diagnosed by angiography or ST elevation criteria.

Figure 1 shows 12-lead ECGs of two cases with similar appearing T-waves in leads V2 and V3, but in the first ECG (A), those T-waves are hyperacute and due to left

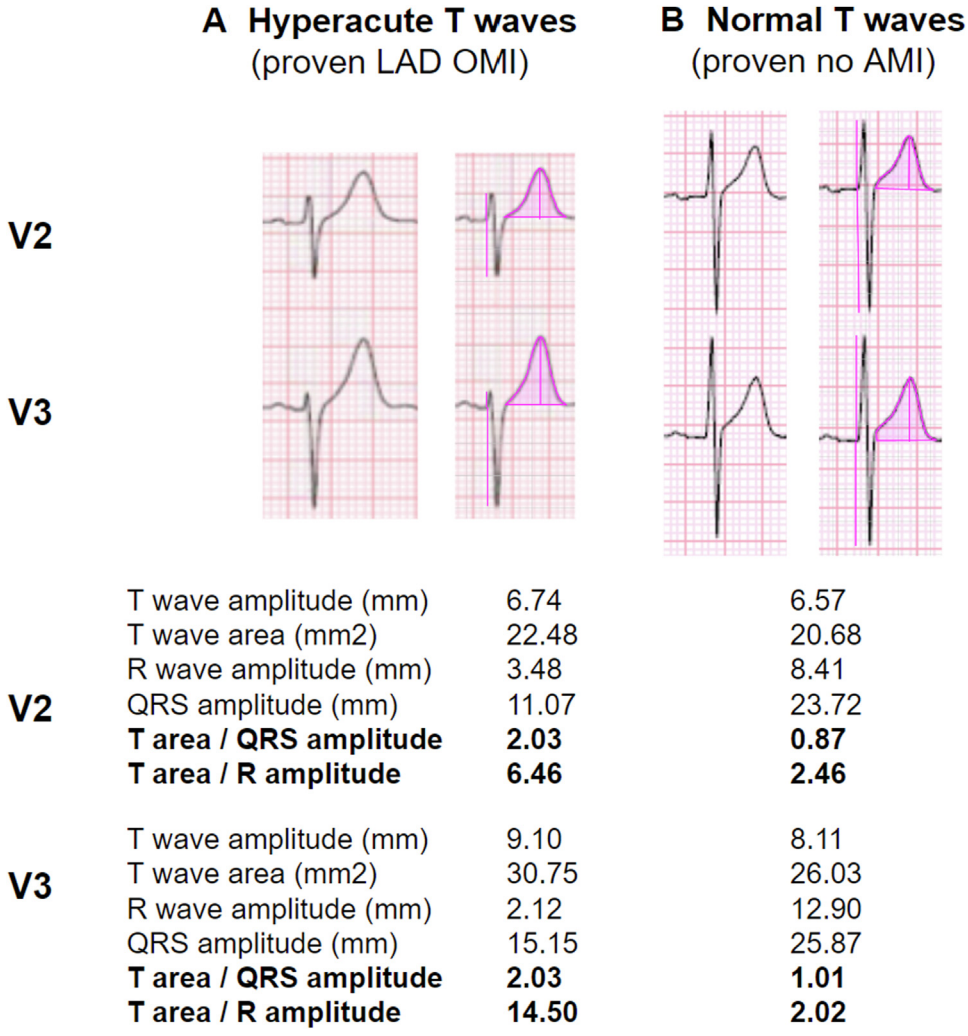

Figure 2. Figure 2 shows measurements of leads V2 and V3 quantifying these characteristics. Thus, hyperacute T-waves when appropriately defined, and interpreted by an expert using that definition, can be an important sign of OMI. *OMI*, occlusion myocardial infarction.

anterior descending coronary artery occlusion, and in the other (B), they are normal. In Figure 2, several measurements of these same T-waves and of the corresponding QRS are made, including the AUC of the T-wave. Then ratios of AUC of the T-wave to QRS and R-wave amplitudes are calculated. These measurements demonstrate that a hyperacute T-wave is not measured only by its amplitude, but by its AUC in relation to the QRS size.

In summary, hyperacute T-waves currently have no formal ECG definition, but expert consensus and existing literature agree that they are likely best defined by increased AUC of the T-wave in proportion to the QRS amplitude (or perhaps QRS area). Like all other ECG findings of occlusion myocardial infarction (such as ST elevation), they should be studied with appropriate outcome definitions; that is, they should be studied for their use in diagnosing patients with acute myocardial infarction who have occlusion myocardial infarction, which needs emergency intervention. Koechlin et al show that T-wave amplitude by itself is not useful in diagnosing undifferentiated acute myocardial infarction. That is not surprising.

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Author affiliations: From the Department of Emergency Medicine (Smith), Hennepin Healthcare and University of Minnesota School of Medicine, Minneapolis, MN; Department of Emergency Medicine (Pendell Meyers), Carolinas Medical Center, Charlotte, NC.

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